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=> S EPITOPE OR DETERMINANT
L2       242750 EPITOPE OR DETERMINANT

=> S L1(w)L2
L3         7868 L1(W) L2

=> S GLUTEN
L4        18870 GLUTEN

=> S PROLAMINE
L5         1030 PROLAMINE

=> S GLIADIN
L6         7330 GLIADIN

=> S L4 OR L5 OR L6
L7        24784 L4 OR L5 OR L6

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L9         7322 L3 NOT 2004/PY

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L11          22 L10 NOT 2003/PY

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=> D L12 1-16 BIB AB

L12  ANSWER 1 OF 16  CA  COPYRIGHT 2004 ACS on STN      DUPLICATE 1
AN   138:38341  CA
TI   Gliadin T Cell Epitope Selection
     by Tissue Transglutaminase in Celiac Disease. Role of enzyme specificity
     and pH influence on the transamidation versus deamidation reactions
AU   Fleckenstein, Burkhard; Molberg, Oyvind; Qiao, Shuo-Wang; Schmid, Dietmar
     G.; von der Mulbe, Florian; Elgstoen, Katja; Jung, Gunther; Sollid, Ludvig
     M.
CS   Rikshospitalet, Institute of Immunology, University of Oslo, Oslo, N-0027,
     Norway
SO   Journal of Biological Chemistry (2002), 277(37), 34109-34116
     CODEN: JBCHA3; ISSN: 0021-9258
PB   American Society for Biochemistry and Molecular Biology
DT   Journal
```

LA English
AB Tissue transglutaminase (TG2) can modify proteins by transamidation or deamidation of specific glutamine residues. TG2 has a major role in the pathogenesis of celiac disease as it is both the target of disease-specific autoantibodies and generates deamidated gliadin peptides that are recognized by CD4+, DQ2-restricted T cells from the celiac lesions. Capillary electrophoresis with fluorescence-labeled gliadin peptides was used to sep. and quantify deamidated and transamidated products. In a competition assay, the affinity of TG2 to a set of overlapping γ-gliadin peptides was measured and compared with their recognition by celiac lesion T cells. Peptides differed considerably in their competition efficiency. Those peptides recognized by intestinal T cell lines showed marked competition indicating them as excellent substrates for TG2. The enzyme fine specificity of TG2 was characterized by synthetic peptide libraries and mass spectrometry. Residues in positions -1, +1, +2, and +3 relative to the targeted glutamine residue influenced the enzyme activity, and proline in position +2 had a particularly pos. effect. The characterized sequence specificity of TG2 explained the variation between peptides as TG2 substrates indicating that the enzyme is involved in the selection of **gluten T cell epitopes**. The enzyme is mainly localized extracellularly in the small intestine where primary amines as substrates for the competing transamidation reaction are present. The deamidation could possibly take place in this compartment as an excess of primary amines did not completely inhibit deamidation of gluten peptides at pH 7.3. However, lowering of the pH decreased the reaction rate of the TG2-catalyzed transamidation, whereas the rate of the deamidation reaction was considerably increased. This suggests that the deamidation of gluten peptides by TG2 more likely takes place in slightly acidic environments.
RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 16 CA COPYRIGHT 2004 ACS on STN DUPLICATE 2
AN 138:152027 CA
TI Celiac lesion T cells recognize epitopes that cluster in regions of gliadins rich in proline residues
AU Arentzp-Hansen, Helene; McAdam, Stephen N.; Molberg, Oyvind; Fleckenstein, Burkhard; Lundin, Knut E. A.; Jorgensen, Thomas J. D.; Jung, Guenther; Roepstorff, Peter; Sollid, Ludvig M.
CS Inst. of Immunol., Univ. of Oslo, Oslo, Norway
SO Gastroenterology (2002), 123(3), 803-809
CODEN: GASTAB; ISSN: 0016-5085
PB W. B. Saunders Co.
DT Journal
LA English
AB Celiac disease is a gluten-induced enteropathy that shows a strong association with HLA-DQ2 and -DQ8. Gluten-specific T cells, invariably restricted by DQ2 or DQ8, can be isolated from celiac lesions. Such gut-derived T cells have a preference for recognition of gluten that has been specifically deaminated by tissue transglutaminase. A systematic characterization of DQ2-restricted **T-cell epitopes** in α- and γ- **gliadins** was conducted. Several new γ-gliadin epitopes and an addnl. α-gliadin epitope were identified by mass spectrometry anal. of peptide fragments of recombinant gliadins and by using synthetic peptides. These epitopes were not randomly scattered across the gliadins but clustered in regions of the proteins with high content of proline residues.
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 16 CA COPYRIGHT 2004 ACS on STN
AN 137:18751 CA
TI Germline mutations in TGM2 do not contribute to coeliac disease

susceptibility in the swedish population
AU Popat, Sanjay; Hogberg, Lotta; McGuire, Susan; Green, Helen; Bevan, Stephen; Stenhammar, Lars; Houlston, Richard S.
CS Section of Cancer Genetics, Institute for Cancer Research, Surrey, SM2 5NG, UK
SO European Journal of Gastroenterology & Hepatology (2001), 13(12), 1477-1479
CODEN: EJGHES; ISSN: 0954-691X
PB Lippincott Williams & Wilkins
DT Journal
LA English
AB Celiac disease (CD) shows a strong genetic predisposition involving HLA-DQ2 and non-HLA components. Tissue transglutaminase, encoded by TGM2, occupies a central role in the CD pathogenesis, necessary for the deamidation of specific glutamine residues of α- **gliadin** creating a **T-cell epitope** that binds with increased affinity to DQ2. To investigate whether germline mutations in TGM2 contribute to disease susceptibility we have carried out a comprehensive anal. of the gene in 52 patients with CD. Blood samples were collected from 52 children with biopsy proven CD attending one Swedish center. DNA was extracted from lymphocytes and all exons and intron-exon boundaries of the TGM2 gene and the alternatively spliced form of the gene were screened for mutations. Mutational anal. was undertaken by a combination of conformational specific gel electrophoresis and direct sequencing. 3 Novel polymorphisms were identified but no pathogenic mutations were detected. There is no evidence from this study that mutations in TGM2, which lead to an altered protein, contribute to CD susceptibility.
RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 16 CA COPYRIGHT 2004 ACS on STN
AN 138:23209 CA
TI Update on immunologic basis of celiac disease
AU Guandalini, Stefano; Gokhale, Ranjana
CS Department of Pediatrics, Section of Gastroenterology, Hepatology and Nutrition, University of Chicago, Chicago, IL, USA
SO Current Opinion in Gastroenterology (2001), 17(6), 545-550
CODEN: COGAEK; ISSN: 0267-1379
PB Lippincott Williams & Wilkins
DT Journal; General Review
LA English
AB A review. During the past few years several seminal studies have greatly expanded our knowledge on celiac disease pathogenesis. This review focuses on aspects that have been most properly addressed and where substantial new information has been gathered include. Topics covered include (a) the identification of **T-cell epitopes** in **gluten** and the mechanisms of specific T-cell response in celiac disease small intestine; (b) the mechanisms of induction of mucosal lesion; and (c) the putative role of non-T-cell factors in driving mucosal response to gliadin. After discussing a brief history of the "quest for the cause of celiac disease," we examine the development of the typical celiac lesion (the crypt hyperplastic mucosal atrophy) as it generally unfolds: the increased entry of dietary antigens; the early changes, linked to specific components of the innate immunity rather than to its adaptive branch; the most thoroughly investigated subsequent response, involving a strong T-cell response and cytokines; and the factors responsible for enterocytes' death. The emerging pattern is that of a complex interaction of factors, although far from being completely understood, but fascinating as it opens an incredible window of knowledge on an autoimmune disorder whose environmental factor is known, whose autoantigen is known, whose autoantibodies are known: a truly unique situation in medicine.

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RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2002:222073 BIOSIS
DN PREV200200222073
TI Bioactivity of peptides homologous to the coeliac disease-specific dominant A-**gliadin T cell epitope.**
AU Anderson, Robert P. [Reprint author]; Jewell, Derek P. [Reprint author]; Hill, Adrian V. [Reprint author]
CS Nuffield Dept of Medicine, Oxford, UK
SO Gastroenterology, (April, 2001) Vol. 120, No. 5 Supplement 1, pp. A.683. print.
Meeting Info.: 102nd Annual Meeting of the American Gastroenterological Association and Digestive Disease Week. Atlanta, Georgia, USA. May 20-23, 2001. American Gastroenterological Association; American Association for the Study of Liver Diseases; American Society for Gastrointestinal Endoscopy; Society for Surgery of the Alimentary Tract.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 3 Apr 2002
Last Updated on STN: 3 Apr 2002

L12 ANSWER 6 OF 16 CA COPYRIGHT 2004 ACS on STN DUPLICATE 3
AN 134:52034 CA
TI Human Genome Search in Celiac Disease Using Gliadin cDNA as Probe
AU Kumar, Rajesh; Lumsden, Angela; Ciclitira, Paul J.; Ellis, H. Julia; Laurie, Gordon W.
CS Department of Cell Biology, University of Virginia, Health Sciences Center, Charlottesville, VA, 22908, USA
SO Journal of Molecular Biology (2000), 300(5), 1155-1167
CODEN: JMOBAK; ISSN: 0022-2836
PB Academic Press
DT Journal
LA English
AB Celiac disease is a wheat gliadin-promoted disorder that displays a complex genetic susceptibility associated with HLA-DQ2, and one or more unknown factor(s), possibly gliadin-like. The presence of mammalian proteins with partial gliadin similarity was suggested by transglutaminase-independent multi-tissue reactivity of gliadin-immunopurified antibodies from celiac patients. No non-plant sequence, however, was identified in gliadin peptide epitope searches of non-redundant and EST databanks via TBLASTN, BLASTP and FASTA, even at E values as high as 20. Therefore, an α-gliadin cDNA screen of human cDNA and genomic libraries was undertaken, an approach in keeping with pos. human Northern and Southern analyses with the same probe. Four distinct cDNA clones were obtained, the most stringent of which (3.2 and 5.1 kb) were novel, and featured potential open reading frames with high gliadin domain II and domain IV homologies (BestFit quality scores ≥295 and 322, resp., vs. random value 126-127). Both were also homologous to ESTs. An addnl. 5' gliadin oligonucleotide screen identified the widely distributed cytoplasmic protein acyl coA hydrolase whose homol. was restricted to the oligonucleotide probe (BestFit quality=215 vs. 100 for random); and achaete-scute homologous protein, which displays particularly high gliadin domain II homol. (BestFit quality 316 vs. 111 for random). Genomic screening uncovered 16 positives, one of which was the ALR gene, whose similarity to three of gliadin's five domains (I, II and IV; BestFit quality 322-473 vs. 121-154 for random) was remarkable. More extensive was novel genomic clone 2, with fragments hybridizing to cDNA probes approximating gliadin domains I, II+IV, V and

the gliadin 5' untranslated region. Genomic clone 2 was mapped by FISH to 19q13.11-13.12. Two fragments were sequenced; one was exonic, as predicted by four different programs; and test oligonucleotides suggested widespread 4 and/or 2 kb mRNA expression, even at high stringency (tm-8.8 deg. C). Taken together, it is apparent that several genes with partial gliadin homol. exist in the human genome. Many of these genes encode proteins that: (1) bear **gliadin**-like **T-cell epitopes**; (2) are expressed in intestine and, (3) like transglutaminase, are cytoplasmic. Glutamine to glutamic acid or other mutation within such epitopes followed by injury or infection-related release could explain enhanced disease susceptibility in affected families. (c) 2000 Academic Press.

RE.CNT 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

ref C on 1449

L12 ANSWER 7 OF 16 CA COPYRIGHT 2004 ACS on STN DUPLICATE 4
AN 132:307160 CA
TI In vivo antigen challenge in celiac disease identifies a single transglutaminase-modified peptide as the dominant A-**gliadin T-cell epitope**
AU Anderson, Robert P.; Degano, Pilar; Godkin, Andrew J.; Jewell, Derek P.; Hill, Adrian V. S.
CS Institute of Molecular Medicine and Gastroenterology Unit, Nuffield Department of Medicine, John Radcliffe Hospital, University of Oxford, Oxford, OX3 9DU, UK
SO Nature Medicine (New York) (2000), 6(3), 337-345
CODEN: NAMEFI; ISSN: 1078-8956
PB Nature America
DT Journal
LA English
AB Celiac disease (CD) is an increasingly diagnosed enteropathy (prevalence, 1:200-1:300) that is induced by dietary exposure to wheat gliadins (as well as related proteins in rye and barley) and is strongly associated with HLA-DQ2 (α1*0501, β1*0201), which is present in over 90% of CD patients. Because a variety of gliadin peptides have been identified as epitopes for gliadin-specific T-cell clones and as bioactive sequences in feeding studies and in ex vivo CD intestinal biopsy challenge, it has been unclear whether a "dominant" T-cell epitope is associated with CD. Here, the authors used fresh peripheral blood lymphocytes from individual subjects undergoing short-term antigen challenge and tissue transglutaminase-treated, overlapping synthetic peptides spanning A-gliadin to demonstrate a transient, disease-specific, DQ2-restricted, CD4 T-cell response to a single dominant epitope. Optimal gamma interferon release in an ELISPOT assay was elicited by a 17-amino-acid peptide corresponding to the partially deamidated peptide of A-gliadin amino acids 57-73 (Q65E). Consistent with earlier reports indicating that host tissue transglutaminase modification of gliadin enhances gliadin-specific CD T-cell responses, tissue transglutaminase specifically deamidated Q65 in the peptide of A-gliadin amino acids 56-75. Discovery of this dominant epitope may allow development of antigen-specific immunotherapy for CD.

RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L12 ANSWER 8 OF 16 CA COPYRIGHT 2004 ACS on STN
AN 133:118581 CA
TI Molecular basis of celiac disease
AU Sollid, Ludvig M.
CS Institute of Immunology, Rikshospitalet, University of Oslo, Oslo, N-0027, Norway
SO Annual Review of Immunology (2000), 18, 53-81
CODEN: ARIMDU; ISSN: 0732-0582
PB Annual Reviews Inc.
DT Journal; General Review

LA English
AB A review and discussion with 145 refs. Celiac disease (CD) is an intestinal disorder with multifactorial etiol. HLA and non-HLA genes together with gluten and possibly addnl. environmental factors are involved in disease development. Evidence suggests that CD4+ T cells are central in controlling an immune response to gluten that causes the immunopathol., but the actual mechanisms responsible for the tissue damage are as yet only partly characterized. CD provides a good model for HLA-associated diseases, and insight into the mechanism of this disease may well shed light on oral tolerance in humans. The primary HLA association in the majority of CD patients is with DQ2 and in the minority of patients with DQ8. Gluten-reactive T cells can be isolated from small intestinal biopsies of celiac patients but not of non-celiac controls. DQ2 or DQ8, but not other HLA mols. carried by patients, are the predominant restriction elements for these T cells. Lesion-derived T cells predominantly recognize deamidated gluten peptides. A number of distinct **T cell epitopes** within **gluten** exist. DQ2 and DQ8 bind the epitopes so that the glutamic acid residues created by deamidation are accommodated in pockets that have a preference for neg. charged side chains. Evidence indicates that deamidation in vivo is mediated by the enzyme tissue transglutaminase (tTG). Notably, tTG can also cross-link glutamine residues of peptides to lysine residues in other proteins including tTG itself. This may result in the formation of complexes of gluten-tTG. These complexes may permit gluten-reactive T cells to provide help to tTG-specific B cells by a mechanism of intramol. help, thereby explaining the occurrence of gluten-dependent tTG autoantibodies that is a characteristic feature of active CD.
RE.CNT 145 THERE ARE 145 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 9 OF 16 CA COPYRIGHT 2004 ACS on STN DUPLICATE 5
AN 133:73198 CA
TI Production of a panel of recombinant gliadins for the characterization of T cell reactivity in coeliac disease
AU Arentz-Hansen, E. H.; McAdam, S. N.; Molberg, O.; Kristiansen, C.; Sollid, L. M.
CS Institute of Immunology, University of Oslo, Oslo, 0027, Norway
SO Gut (2000), 46(1), 46-51
CODEN: GUTTAK; ISSN: 0017-5749
PB BMJ Publishing Group
DT Journal
LA English
AB Background/Aims-Coeliac disease is a chronic intestinal disorder most probably caused by an abnormal immune reaction to wheat gliadin. The identification of the HLA-DQ2 and HLA-DQ8 as the mols. responsible for the HLA association in coeliac disease strongly implicates a role for CD4 T cells in disease pathogenesis. Indeed, CD4 T cells specific for gliadin have been isolated from the small intestine of patients with coeliac disease. However, identification of **T cell epitopes** within **gliadin** has been hampered by the heterogeneous nature of the gliadin antigen. To aid the characterization of **gliadin T cell epitopes**, multiple recombinant **gliadins** have been produced from a com. Nordic wheat cultivar. Methods-The α-gliadin and γ-gliadin genes were amplified by polymerase chain reaction from cDNA and genomic DNA, cloned into a pET expression vector, and sequenced. Genes encoding mature gliadins were expressed in Escherichia coli and tested for recognition by T cells. Results-In total, 16 α-gliadin genes with complete open reading frames were sequenced. These genes encoded 11 distinct gliadin proteins, only one of which was found in the Swiss-Prot database. Expression of these gliadin genes produced a panel of recombinant α-gliadin proteins of purity suitable for use as an antigen for T cell stimulation. Conclusion-This study provides an insight into the complexity of the

<post dates British priority>

gliadin antigen present in a wheat strain and has defined a panel of pure gliadin antigens that should prove invaluable for the future mapping of epitopes recognized by intestinal T cells in coeliac disease.

RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 10 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2000:230593 BIOSIS
DN PREV200000230593
TI Gluten challenge in coeliac disease reveals a single transglutaminase-modified peptide as the dominant **T cell epitope** in A-**gliadin**.
AU Anderson, R. P. [Reprint author]; Degano, P. [Reprint author]; Godkin, A. J. [Reprint author]; Jewell, D. P. [Reprint author]; Hill, A. V. S. [Reprint author]
CS Nuffield Department of Medicine, Institute of Molecular Medicine, University of Oxford, John Radcliffe Hospital, Oxford, OX3 9DU, UK
SO Gut, (April, 2000) Vol. 46, No. 11, pp. A33. print.
Meeting Info.: 2000 Annual Meeting of the British Society of Gastroenterology. Birmingham, UK. March 21-23, 2000. British Society of Gastroenterology.
CODEN: GUTTAK. ISSN: 0017-5749.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 7 Jun 2000
Last Updated on STN: 5 Jan 2002

not of record post dates British priority date

L12 ANSWER 11 OF 16 CA COPYRIGHT 2004 ACS on STN
AN 130:251211 CA
TI Peptides specific for gluten-sensitive T-cells and use thereof
IN Koning, Frits; Van De Wal, Yvonne; Drijfhout, Jan Wouter; Kooy-Winkelaar, Engelina Maria Christina
PA Academisch Ziekenhuis Leiden, Neth.; Rijksuniversiteit Leiden
SO Eur. Pat. Appl., 58 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 905518	A1	19990331	EP 1997-202909	19970923
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI EP 1997-202909		19970923		

ref A on 1449

AB The invention relates to the field of immunol., more specifically to food-related immune enteropathies (gluten-sensitivities) such as celiac sprue, tropical sprue, giardiasis and food allergies of childhood, but also to disorders such as dermatitis herpetiformis (DH). The invention provides a method to find or characterize peptides that are recognized by intestinally derived gluten-specific T-cell which is instrumental in gluten sensitivity. The invention also provides such peptides which can be obtained from a prolamine, such as a gliadin, secalin, hordein, avenin and glutenins and provides peptides constituting a **T-cell epitope** obtainable from **gliadin** and glutenin, for example comprising the sequence SGQGSFQPSQQ or GQQGYYPTSPQQSGQ or derivs. thereof having similar properties.

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 12 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

AN 1999:330652 BIOSIS
DN PREV199900330652
TI Identification of a coeliac disease-specific **T cell epitope** from A-**gliadin**.
AU Godkin, A. [Reprint author]; Brookes, R. [Reprint author]; Jewell, D. P. [Reprint author]
CS Radcliffe Hosp, Oxford, UK
SO Gastroenterology, (April, 1999) Vol. 116, No. 4 PART 2, pp. A882. print.
Meeting Info.: Digestive Disease Week and the 100th Annual Meeting of the American Gastroenterological Association. Orlando, Florida, USA. May 16-19, 1999. American Gastroenterological Association.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 24 Aug 1999
Last Updated on STN: 24 Aug 1999

L12 ANSWER 13 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1999:214004 BIOSIS
DN PREV199900214004
TI Identification of a coeliac disease-specific **T cell epitope** from A-**gliadin**.
AU Godkin, A. J. [Reprint author]; Brookes, R. [Reprint author]; Jewell, D. P.; Hill, A.V.S. [Reprint author]
CS Molecular Immunology Group, Institute Of Molecular Medicine, John Radcliffe Hospital, Oxford, UK
SO Gut, (April, 1999) Vol. 44, No. SUPPL. 1, pp. A72. print.
Meeting Info.: British Society of Gastroenterology Annual Meeting. Glasgow, Scotland, UK. March 23-25, 1999. British Society of Gastroenterology.
CODEN: GUTTAK. ISSN: 0017-5749.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 26 May 1999
Last Updated on STN: 26 May 1999

ref 6 of 1449

L12 ANSWER 14 OF 16 CA COPYRIGHT 2004 ACS on STN
AN 129:188106 CA
TI Use of complete eluted peptide sequence data from HLA-DR and -DQ molecules to predict T cell epitopes, and the influence of the nonbinding terminal regions of ligands in epitope selection
AU Godkin, Andrew J.; Davenport, Miles P.; Willis, Anthony; Jewell, Derek P.; Hill, Adrian V. S.
CS Mol. Immunology Group, Inst. of Mol. Medicine, John Radcliffe Hospital, Oxford, UK
SO Journal of Immunology (1998), 161(2), 850-858
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
AB In diseases with a strong association with an HLA haplotype, identification of relevant T cell epitopes may allow alteration of the pathol. process. In this report the authors use a reverse immunogenetic approach to predict possible HLA class II-restricted T cell epitopes by using complete pool sequencing data. Data from HLA-DR2 (B1*1501), -DR3 (B1*0301), -DQ2 (A1*0501, B1*0201), and -DQ8 (A1*0301, B1*0302) alleles were used by a computer program that searches a candidate protein to predict ligands with a relatively high probability of being processed and presented. This approach successfully identified both known T cell epitopes and eluted single peptides from the parent protein. Furthermore, the program

identified ligands from proteins in which the binding motif of the HLA mol. was unable to do so. When the information from the non-binding N- and C-terminal regions in the pool sequence was removed, the ability to predict several ligands was markedly reduced, particularly for the HLA-DQ alleles. This suggests a possible role for these regions in determining ligands
for HLA class II mols. Thus, the use of complete eluted peptide sequence data offers a powerful approach to the prediction of HLA-DQ and -DR peptide ligands and T cell epitopes.
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 15 OF 16 CA COPYRIGHT 2004 ACS on STN DUPLICATE 6
AN 129:314913 CA
TI Identification of a **gliadin T-cell epitope** in celiac disease: general importance of gliadin deamidation for intestinal T-cell recognition
AU Sjostrom, H.; Lundin, K. E. A.; Molberg, O.; Korner, R.; McAdam, S. N.; Anthonsen, D.; Quarsten, H.; Noren, O.; Roepstorff, P.; Thorsby, E.; Sollid, L. M.
CS Department of Medical Biochemistry and Genetics, Biochemistry Laboratory C, The Panum Institute, University of Copenhagen, Copenhagen N, DK-2200, Den.
SO Scandinavian Journal of Immunology (1998), 48(2), 111-115
CODEN: SJIMAX; ISSN: 0300-9475
PB Blackwell Science Ltd.
DT Journal
LA English
AB Celiac disease probably results from a T-cell response to wheat gliadin and is associated to HLA-DQ2. No gliadin epitopes recognized by intestinal T cells have yet been identified, limiting the understanding of the pathogenesis. Gut lesion-derived DQ2-restricted T cells from celiac disease patients were used to identify an epitope within a purified γ-type gliadin. The structure of the epitope was characterized by mass spectrometry and verified by synthesis. The epitope (QPQQSFPEQQ) results from deamidation of a distinct glutamine in the native structure. This deamidation is important for binding to DQ2 and T-cell recognition. Other gut-derived T cells fail to recognize the epitope, although deamidation of unfractionated gliadin enhances the response of all gut-derived DQ2-restricted T cells isolated from several patients. Several DQ2-restricted T-cell epitopes exist, but for all of them deamidation of glutamine residues appears to be critical for creation of active epitopes. Native gliadin has few neg. charged residues but is very rich in glutamine. After deamidation gliadin becomes a rich source of DQ2 epitopes thus providing a link between DQ2, gliadin, and celiac disease. The necessity for modification may have general immunol. relevance.
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

does not appear to be relevant

L12 ANSWER 16 OF 16 CA COPYRIGHT 2004 ACS on STN
AN 128:153217 CA
TI The molecular basis of the HLA association in celiac disease
AU Sollid, L. M.; Johansen, B. H.; Lundin, K. E. A.; Molberg, O.; Scott, H.; Vartdal, F.; Thorsby, E.
CS Institute of Transplantation Immunology The National Hospital, University of Oslo, Oslo, Norway
SO NATO ASI Series, Series 3: High Technology (1997), 35(Immunogenetics: Advances and Education), 61-69
CODEN: NAHTF4; ISSN: 1383-7168
PB Kluwer Academic Publishers
DT Journal; General Review
LA English
AB A review with 23 refs. Discussed are: the HLA association in celiac disease

(CD); isolation of gluten-specific T cells from the small intestine; HLA restriction of gluten-specific T cells; antigen specificity of the gluten-specific T cells; the peptide binding motif of DQ(α1*0501,β1*0201); an attempt to predict a DQ2-restricted **gliadin T cell epitope**; possible important steps in the development of CD; and type 1 diabetes and other HLA-associated diseases (lessons from CD).

RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D HIS

(FILE 'HOME' ENTERED AT 10:06:50 ON 26 NOV 2004)

FILE 'CA, BIOSIS' ENTERED AT 10:06:59 ON 26 NOV 2004

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L1      406349 S T(W)(CELL OR LYMPHOCYTE)
L2      242750 S EPITOPE OR DETERMINANT
L3        7868 S L1(W)L2
L4       18870 S GLUTEN
L5        1030 S PROLAMINE
L6        7330 S GLIADIN
L7       24784 S L4 OR L5 OR L6
L8          30 S L3(5A)L7
L9        7322 S L3 NOT 2004/PY
L10         27 S L8 NOT 2004/PY
L11         22 S L10 NOT 2003/PY
L12         16 DUPLICATE REMOVE L11 (6 DUPLICATES REMOVED)
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